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Recent Research in Behind-Armor Blunt Trauma and Traumatic Brain Injury

by Wendy E. Bukowski

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The occurrence of traumatic brain injury (TBI) among U.S. Soldiers is daunting. Therefore, the ongoing effort to develop more advanced technologies for Soldier protection requires a complete understanding of the work that has been done. Although a broad range of fields of expertise have looked at TBI, there remains little understanding of the TBI mechanism. Also of interest is the role of the helmet and its effectiveness against blunt impact and blast threats. Computational modeling can offer great insight into the injury mechanisms of the brain due to blunt force trauma. These predictive models can help mold the direction of helmet research and development. This report gives a broad overview of the work that has been done in the area of behind-armor blunt trauma and traumatic brain injury research.

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1. Introduction

The occurrence of traumatic brain injury (TBI) among U.S. Soldiers is daunting. Therefore, the ongoing effort to develop more advanced technologies for Soldier protection requires a complete understanding of the work that has been done. Although a broad range of fields of expertise have looked at TBI, there remains little understanding of the TBI mechanism. Additionally of interest is the role of the helmet and its effectiveness against blunt impact and blast threats.

Computational modeling can offer great insight into the injury mechanisms of the brain due to blunt force trauma. These predictive models can help mold the direction of helmet research and development.

This paper gives a broad overview of the work that has been done in the area of behind-armor blunt trauma and traumatic brain injury research.

2. Research and Development

2.1 Loading Conditions

Traumatic brain injuries can occur as a result of various loading conditions, including automobile accidents, blunt trauma, blast from explosive devices, and pressure waves generated by explosions. An improved understanding of the mechanisms by which these loading conditions translate into brain deformation and damage is necessary to gain better insights into the observed health consequences associated with TBI.

Bass et al. (*1*) investigated brain injuries resulting from explosive blast and identified specific sources of mechanical insult, each with different characteristics; for example, blast-wave impingement where direct effects of blast-induced shock and the overpressure wave on the body are seen, fragment penetration resulting from any projectile energized by the blast that penetrates the skin, and blunt trauma resulting from the body being thrown through space into a structure such as a building, wall, or the ground, as well as falls and impact by high-rate projectiles (nonpenetrating), either directly or behind protective equipment. Scaled animal models were used to conclude that fatality from neurotrauma occurs only at higher exposures than is necessary to cause fatalities from pulmonary consequences in unprotected humans. But when a gyrencephalic animal model was studied, there was evidence of mild brain injuries at blast intensities that are similar to the pulmonary injury threshold (*1*).

For the animal models to be accurately correlated to human response, consideration of the experimental model is important to appropriately simulate realistic but repeatable blast shocks, especially for scaling. Actual high-explosive or shock-tube experiments, including location of the animal model relative to the shock tube, should be carefully designed to provide a realistic blast experiment with conditions comparable to blasts on humans.

Finite element (FE) modeling is well suited for studying the mechanical response of the head to primary blast loading, and researchers have recently developed FE models to improve our understanding of blast TBI (*2*–*5*). A major limitation of some current blast models is that their loading conditions are not representative of real-world blast events. Many FE models simulate exposures based on small charge sizes (less than 1-kg TNT) at a very close standoff (less than 1 m), which may reproduce realistic peak overpressure but very low positive-phase duration or impulse (*3*, *6*, *7*).

Models that do simulate real-world blast events consider only a single blast condition, making it difficult to assess the biomechanics that may cause injury from blast impact. Rafaels et al. (*8*) used animal models in their research to suggest that blast TBI may also depend on peak overpressure and duration, implying that computational models need to simulate a wide range of blast scenarios to understand the potential injury mechanisms.

Panzer et al. (*9*) developed a detailed model of the human head for simulating the brain's response to a wide range of real-world threats. Since a robust set of experimental data was not available for validating any human head model in blast, it was more practical to develop an exploratory plane-strain blast model (figure 1) before attempting to develop a complex three-dimensional (3-D) head model. They examined many of the important aspects of numerical blast modeling in detail using this model, where it is often not feasible on a more complicated model.

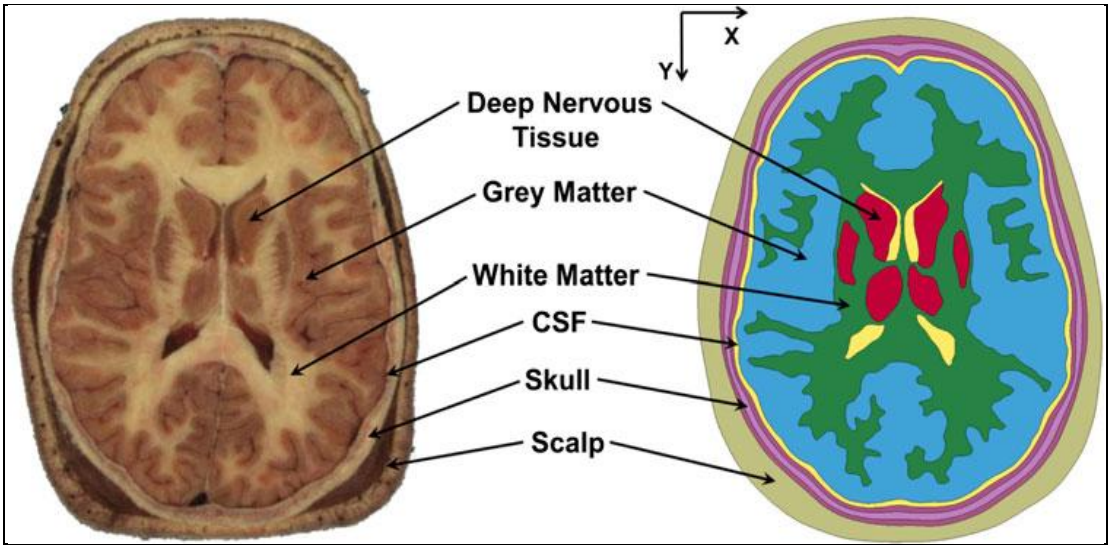

Figure 1. Brain geometry from photos: (left) Visible Human Project and (right) plane-strain model (*9*).

Figure 2 shows the peak incident overpressure and duration conditions for blast simulation compared with human primary blast injury criteria and real-world blast threats. Eighteen cases were simulated with varying blast waves that produced peak incident overpressure values 50–100 kPa and 1–8 ms of positive phase duration. These data were compared with previously reported peak overpressure-duration characteristics that are commonly seen in combat (*3*, *8–11*). Additionally, data for two commonly used munitions were calculated using ConWep calculations to serve as a real-world reference to the simulated blast conditions (*12*).

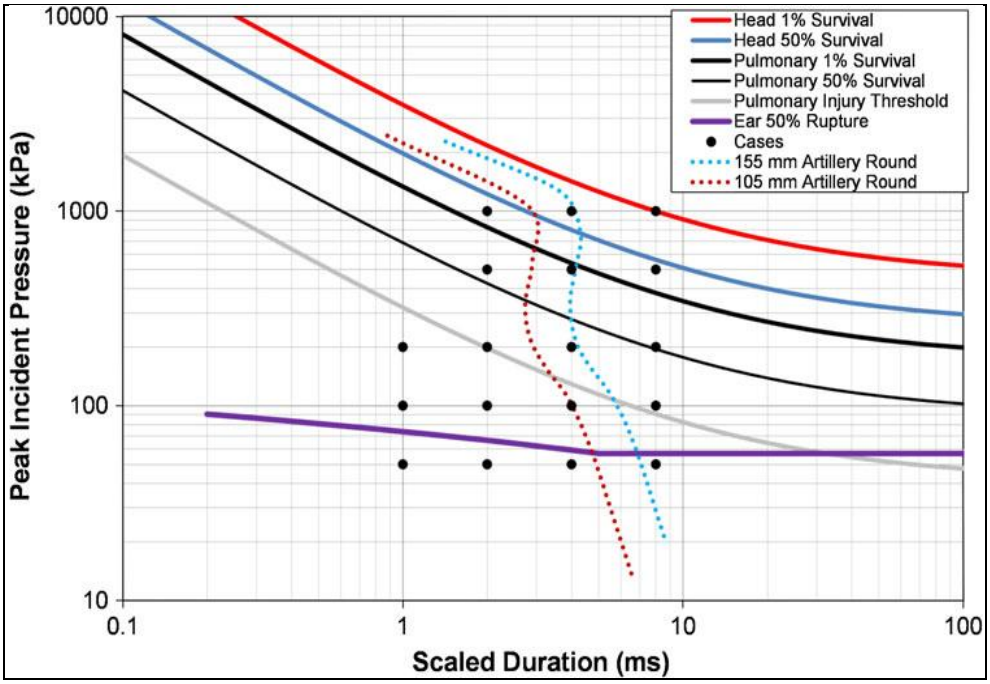

Figure 2. Peak incident overpressure and duration conditions for blast simulation compared with human primary blast injury criteria and real-world blast threats (*9*).

Over the wide range of blast conditions simulated, the maximum pressure levels within the brain were primarily dependent on the overpressure conditions of the blast wave. The insensitivity of pressure response to the blast duration was likely because the speed of sound in the skull and brain were much quicker than that of air. The higher sound speed propagated pressure away from the impact site faster than it was loaded (*9*).

2.3 Type of Injuries

Magnuson et al. (*13*) define TBI from explosive blast as either closed head (cTBI) or penetrating TBI (pTBI). They describe mild TBI (bTBI) as being characterized by an initial period of abnormal awareness, then ranging from confusion to brief loss of consciousness lasting a few minutes or less. Moderate to severe bTBI occurs when there is gross structural brain damage from the explosive blast. Patients present symptoms of an altered mental status, ranging from confusion to lethargy to coma. Neuroimaging is rare but can reveal intracranial hemorrhage, skull fracture, cerebral edema, and parenchymal contusions. Diffusion tensor imaging reveals

dose-dependent diffuse axonal injury (DAI), where the magnitude of injury depends on the extent of rotational movement of the brain, is different from concussive impact DAI. Brains receiving a bTBI can develop malignant cerebral edema very quickly, on the order of an hour or so, as opposed to several hours to a day following cTBI (*13*).

There are a variety of side effects that can be attributed to mild explosive blast TBI, such as headache, confusion, amnesia, difficulty concentrating, short-term memory loss, mood alteration, sleep disturbance, vertigo, and anxiety. Most often these symptoms occur immediately after injury and resolve after a few hours or days.

Diffuse injury to the brain can be a product of explosive blast TBI, due to increase in intracranial pressure from pressure loading, and coup-contrecoup impact with the skull, caused by primary or tertiary injury mechanisms. The transmitted waves or rotational-translational acceleration can also cause diffuse axonal injury due to sheer strain. Local regions of injury in the cortex, white matter tracts, cerebellum, and cerebral ventricles can occur due to the brain colliding against the skull.

The cellular neuropathology of the brain can be affected by explosive blast TBI; for example, an abnormal increase in the number of astrocytes due to the destruction of nearby neurons. Also, the cytoskeletal components of the neuron are susceptible to damage and rearrangement in bTBI, and the oligodendrocytes can go through apoptosis.

Focal brain injuries result from physical impact to the head, coup, or contrecoup, and can be seen as contusions, lacerations, epidural/subdural hemorrhage, and intracerebral/intraventricular hemorrhage (*14*). The development of coup and contrecoup lesions occurs by the rapid increases in acceleration, which leads to the development of increased intracranial pressure at the trauma point and decreased (contrecoup) pressure at the opposite side of the cerebrum. In addition to the brain striking the opposite side of the skull, this decreased pressure at the contrecoup site can cause the development of cavitation bubbles that cause local tissue damage when they burst or collapse.

Diffuse injuries can be caused by the acceleration/deceleration and/or rotation of the brain within the skull. While difficult to detect, these injuries include DAI, ischemic brain injury, and swelling that can lead to increased intracranial pressure. DAI can result in substantial diffuse microscopic damage and intense shearing and stretching of axons throughout the brain and brain stem in addition to focal lesions in the corpus callosum. The twisting strains on the axons cause them to be torn in half (*15*, *16*).

The U.S. Centers for Disease Control and Prevention reports that at least 1.7 million TBIs occur every year either as an isolated injury or along with other injuries (*17*). In addition to the physical consequences of TBI, the economic impact of direct medical costs and indirect costs such as lost productivity totaled an estimated $76.5 billion in the United States in 2000 (*18*). Taylor and Ford (*19*) used numerical simulations to investigate the early time wave interaction in the human head

when impacted by an automobile windshield. They were able to determine that these wave interactions produced significant levels of stress at localized regions within the brain on an early time scale (1 ms) preceding any overall motion of the head. The simulations of the interactions of the skull, brain, and cerebral spinal fluid with the windshield glass showed that there were at least two TBI mechanisms. The first comprises the cell volume changes that occur as a result of compressive and tensile pressures that cause internal cell damage. The second mechanism is the tearing of the cellular membranes of brain neurons that happens with the development of shearing stresses at localized regions of the brain surrounding the ventricles. This state leads to a loss of electrical impulse conductivity and cell functionality. One of the key findings from this research was that there was significant stress generated in the head during the impact but before any overall head motions occurred.

2.4 Theories of Injury Origin

Two proposed injury mechanisms cited by Bass et al. (*1*) include direct transmission of the blast wave across the head/facial structures, which are strongly supported by direct measurements of pressure transmission into the brain, and indirect transmission through the torso into the head through the vasculature of neck primarily proposed by Cernak et al. (*20*).

The blast wave generated from a shock front followed by blast overpressure can have various serious effects on the body. Blast waves impinging on the human body can be reflected, transmitted, or a combination of both. Internal response to blast wave can vary due to the wave moving at variable speeds relative to tissues of different density. This can lead to relative motion between the tissue, resulting in shearing and tearing at the tissue interface (*21*). Blast wave pressure loading and velocity in tissue, variable tissue density, pressure rise-time, and pressure decay are all variables that affect tissue response to explosive blasts. Additionally, the interaction of the blast wave with the human head results in pressure loading of the human head, which has two components: (1) the incident shock front or reflective shock front, depending on the aforementioned reflective waves and (2) pressure due to the slower moving blast wind. Blast-wave impingement on a spherical surface wraps itself around the sphere, resulting in an elevated point of pressure where these waves meet opposite the blast source (*21*). Pressure loading of the skull has been found to cause skull flexure and increases in intracranial pressure (*22*).

Due to similar densities of both brain and cerebral spinal fluid (CSF), the brain normally remains buoyant in the CSF, which behaves like a homogenous viscous liquid. This allows the brain to move within the skull under low levels of translational and rotational acceleration (*23*). The brain slows rigid body displacement at low impact speed, while at higher impact speeds, brain motion is due to deformation (*24*). Since the space between the skull and brain is thin and filled with CSF, any use of excess external forces can cause the skull to collide with the brain or the brain to collide with the skull. The resulting relative motion can produce subdural hematomas through tearing of bridging veins. Intracerebral hematomas may also occur when parenchymal blood

vessels rupture during the collision between the brain and skull. Elkin et al. (*25*) performed stress relaxation tests on porcine brain samples and found that cerebellar grey and white matter, brainstem, and corpus callosum are the softest areas measured while the stiffest areas of the brain included the cortex and the hippocampal CA1/CA3 regions.

Cavitation is the formation of microscopic bubbles within brain tissue as it is pulled away from the skull when the head suddenly stops or accelerates. Cavitation occurs when an object moves rapidly through a liquid, such as when the brain moves through cerebral spinal fluid. The formation and collapse of these bubbles causes disruption of brain tissue. Cavitation injuries generally occur on the opposite side of the brain from the point of impact. They are sometimes referred to as contrecoup injuries.

An important area of interest in FE blast modeling is how CSF cavitation formation and collapse affects the mechanical response of the brain to blast loading. CSF cavitation has long been considered to be a brain injury mechanism for blunt impact and blast, but only a few FE blast brain analyses have included CS cavitation in the model (*26–28*).

Panzer et al. (*9*) determined that the largest pressures measured throughout the majority of the brain were from the initial brain shock wave produced from the blast wave impacting the head. The frontal portion of the brain experienced the highest values of peak brain pressure that corresponds to the blast impact site. Panzer et al. also found that CSF cavitation collapse caused a number of localized high-pressure regions in brain tissues adjacent to the CSF, including the periventricular tissues (figure 3).

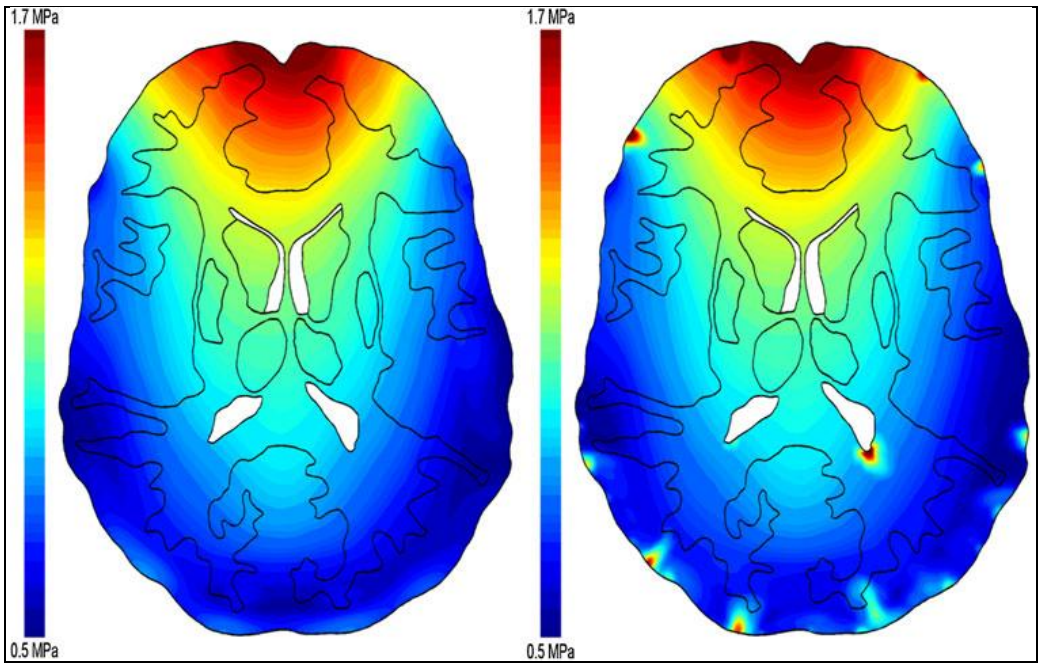

Figure 3. Comparison of the distributions peak brain tissue pressure between the noncavitating (left) and cavitating (right) models for the 500-kPa/4-ms blast condition (*9*).

CSF cavitation had more of an effect on the deformation of the brain than seen with the pressure response. They theorized the presence of cavitation caused the brain tissue to separate from the skull, thus allowing the less-constrained brain material to deform more easily. The largest relative increases in brain tissue strain produced by CSF cavitation were in the occipital region of the brain and the periventricular tissues (*9*).

The deviatoric response of the brain was driven largely by skull deformation and not head motion (i.e., rigid body inertia), confirming previous computational results (*22*). Strain developed mainly in the cerebral cortex as it moved relative to the vibrating skull. Decreasing the radial coupling between the brain and the skull (via CSF cavitation) increased the lag between the brain and skull and augmented strain levels. It is important to consider that maximum stresses and strains occurred after the initial shock wave passed through the head. Many FE models did not run the simulation past 2 ms, capturing only the response of the brain during the incident wave and not the response caused by the coupling between the brain and the deforming skull. If high-rate strain response from skull deformation following blast exposure is the primary mechanism for blast TBI, then it is critical that models are simulated long enough to capture these effects.

CSF cavitation has long been hypothesized as a cause of brain injury in both automotive head impacts and blast impacts. However, it remains unclear whether CSF cavitation occurs during these impact events and at what tensile pressures. CSF cavitation may also play a role in damaging deeper neural tissues. The periventricular tissues experienced higher levels of strain when CSF cavitation occurred in addition to the elevated levels of pressure from cavitation collapse.

Either pressure is not an injury mechanism for brain tissue in blast or a pressure-based injury criterion will include rate effects that would decrease the tissue threshold from the hydrostatic criterion. Maximum pressure levels within the brain were primarily dependent on the overpressure conditions of the blast wave, even in the presence of CSF cavitation, implying that if pressure was the only injury mechanism of brain tissue, then TBI should not depend on blast duration or impulse. This would be unlike short-duration pulmonary blast injury criteria, where injury risk is strongly associated with both peak overpressure and duration.

It is possible that the injury mechanisms responsible for gross injury of brain tissue in blast are not the same as mechanisms that are responsible for mild TBI. Furthermore, it has been previously shown that tissue injury thresholds can vary by region in the brain. The increase in strain caused by CSF cavitation for periventricular tissues may exceed injury thresholds prior to tissue in the cortex, which has the largest predicted strains but also the higher threshold for injury.

There has been ongoing research looking at how the brain shifts inside the skull during an impact event because this is crucial in understanding the head injury mechanism. Earlier work did not have the technological ability to provide 3-D quantitative brain motion data (*29–31*). King et al. (*32*) used high-speed biplane x-ray and neutral density targets to collect 3-D data of brain displacement and deformation in human cadavers. It was observed that as the head begins to

rotate, local brain tissue is inclined to keep its shape and position with respect to the inertial frame, which creates the brain displacement and deformation. Also, as the rotation of the head slows or changes direction, the motion of the brain due to angular rotation is greater than that of the skull. When looking at the effects of linear acceleration, the motion is considerably less, implying that the injury mechanism is quite different and is the result of a dynamic pressure wave that is generated during blunt impact.

Much research has been focused on the blast overpressure that results from head injuries caused by explosive munitions (*33, 34*). Fiskum et al. (*35*) speculated that there are a number of physical factors that are involved as well, such as blast overpressure, thermal and chemical components, shockwave, and hyper-acceleration of the brain. In particular, they hypothesized that the hyper-acceleration combined with the subsequent rapid deceleration of the head could be a mechanism responsible for many aspects of brain injury.

2.5 Role of Protective Gear

The combination of a detailed FE model of the human head and a realistic model of a helmet are crucial in gaining an accurate assessment of injuries to the head caused ballistic impact. This knowledge is important in the development of future ballistic helmet design. Aare and Kleiven (*36*) investigated the effects of helmet shell stiffness and different impact angles on load levels in the human head during impact. The material properties of the helmet shell were altered to obtain the various stiffness values. Using a constant helmet shell configuration, four different impact angles were tested. The stress in the cranial bone, maximum principal strain in the brain tissue, pressure in the brain, change in rotational velocity of the skull, and translational and rotational acceleration of the skull were measured using the finite element method (FEM) in LS-DYNA3D.

Aare and Kleiven found that the difference in the maximum and minimum pressure in the brain was greatest for the most flexible helmet shell, and the more flexible shell caused a marked increase of von Mises stress in the cranial bone compared with the more rigid shell. Additionally, the stress in the cranial bone increased with the impact angle and a larger strain in the brain tissue was seen for the 45° impact than for the 90° impact. The area of failed elements, such as delamination behavior with separation of the solid layers, was seen to increase with decreasing material stiffness and strength. The distinction between injury mechanisms can be seen when the helmet shells are rigid enough to avoid contact between the helmet and skull, resulting in the load being transferred through the pads/suspension where the helmet and skull collide, resulting in a localized indentation of the helmet to the skull. It was determined that the stress in the cranial bone increases as the angle of impact increases, and is further accentuated when the impact angle causes contact between the helmet shell and skull. The results of these experiments showed that the highest strains in the brain tissue were seen for an impact angle of 45° when the impact has a tangential component and the rotational effects are transferred to the head (*36*). Note that the results of this study involved impact at only one location, the forehead region, which is known to be able to withstand more force than other areas of the head (*37*).

The purpose of a combat helmet is to attenuate the shock by absorbing the energy generated during impact through deformation and dissipating the energy rapidly through deformation of the helmet and padding material to spread the ballistic loads over a larger area and longer time. Despite the technological advancements of modern combat helmets in preventing bullets of handguns and some rifles from penetrating them, traumatic injuries to both the brain and skull can still occur due to the excessive mechanical responses of the helmet and the head. A ballistic impact on a helmet can generate peak head acceleration that may exceed the tolerance level of what the tissues can withstand, causing irreversible damage to these tissues. Head injuries can also be found when the bullet has sufficient energy to cause the interior helmet shell to come in contact with the underlying tissue, known as "rear effect" (*38*).

Testing of helmets to determine the injuries to the skull and brain have been a challenge for a number of reasons. There is a great deal of variability in the tests that are done by different helmet manufacturers that make comparisons difficult. Also, the materials used to simulate the human head can vary, which results in different injury outcomes. Additionally, these tests can be costly and lengthy. The use of numerical models has offered a more cost-effective method to evaluate the helmet protection than experimental tests. These numerical simulations, such as the FEM, offer an understanding of the mechanism head injury during impact and how the helmet accentuates these injuries. Tan et al. (*39*) were able to show good correlation of results between FE modeling experiments and experimental impact tests of the performance of the Advanced Combat Helmet (ACH). They also obtained similar FE simulation and experimental results for the effectiveness of the ACH interior cushioning system. It was determined that the use of softer foam padding with lower stiffness is more efficient as shock absorbing materials against ballistic impacts.

Chu et al. (*40*) developed a custom sensor to be used in the helmet for the measurement of over-pressurization and blunt impact that will allow for the distinction between primary, secondary, tertiary, and quaternary effects. This sensor was intended to be used in the battlefield to measure the true blast exposure to the Soldier. Previous efforts (*41*) have involved mounting a sensor on the helmet, which has many drawbacks including the inability to differentiate the helmet acceleration from the head acceleration. The effects of human exposure to blast are critical in assessing the source of the injury associated with over-pressurization and blunt impact.

2.6 Research Gaps

There are no well-developed tools to estimate primary blast brain injury risk in a manner similar to those used for pulmonary blast risk assessments. Such injury risk assessments are essential in determining the potential for mild TBI from blast, guiding experimentation, and improving the design of new or existing equipment used to mitigate blast exposures.

El Sayed et al. (*42*) studied the biomechanical modeling of the brain tissue response to traveling impact waves and the computational simulation of traumatic brain injuries. FEM has become a powerful tool for studying mechanics of brain injury. While biomechanical modeling of

traumatic brain injuries requires the formulation of complex constitutive equations, accounting for large strains, time and rate effects, and consistent damage models, the development of such a model has been a challenge due to the geometric complexity of the human head, material compositions, boundary/interface conditions, as well as insufficient experimental data for model validation.

From exterior to interior, the human head consists mainly of scalp, skull, membranes (dura, arachnoid, and pia mater), CSF, and brain. Each component has its own unique structure and complex geometry. For example, the skull is a three-layer structure consisting of an inner and an outer table of cortical bones and an inner layer of cancellous bone.

Furthermore, the skull bone varies significantly in its thickness from location to location. The brain is known to be one of the most complex biological structures. Its tissues can be divided into two types: gray matter, made up mainly of the cell bodies of neurons, and white matter, primarily comprising the axons of neurons. Apart from the complex geometry, biological tissues are often inhomogeneous, anisotropic, nonlinear, and their well-defined mechanical properties are still lacking. In addition, interactions between different material compositions such as solid skull, gel-like brain, and fluids pose additional challenges in FEM. Finally, head injury experiments are difficult and expensive to carry out, and very limited data, especially, experimental data on in vivo human brains, are available for FEM validation.

3. Summary

There has been extensive research in a variety of aspects that contribute to TBI to further understand the phenomena and therefore develop mitigation steps to prevent these injuries. A comprehensive understanding of how Soldier protection is used and areas in which need improvement require ongoing research for the ever-changing environments our Soldiers encounter.

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List of Symbols, Abbreviations, and Acronyms

TBI	traumatic brain injury
cTBI	closed-head TBI
pTBI	penetrating TBI
bTBI	blast-induced mild TBI
DAI	diffuse axonal injury
CSF	cerebral spinal fluid
FEM	finite element model
ACH	Advanced Combat Helmet

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